Kazrin binds periplakin

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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Literature references

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Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

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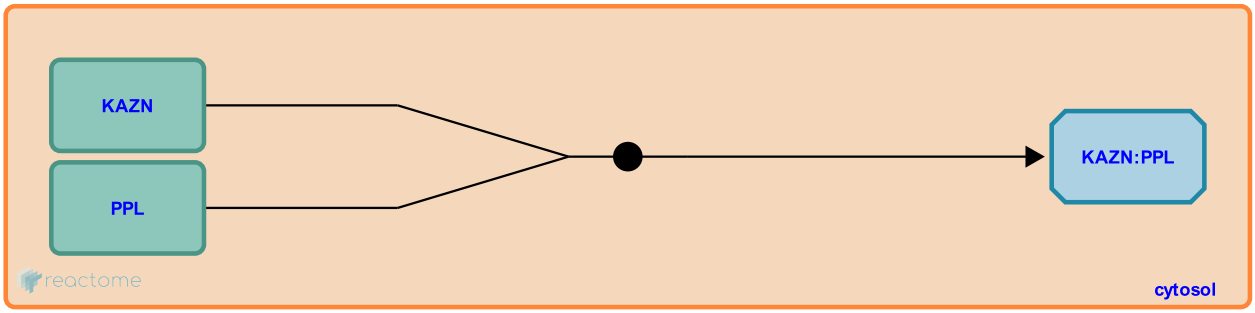
Stable identifier: R-HSA-6814374

Type: binding

Compartments: cytosol



Kazrin (KAZN) is an evolutionarily-conserved cytoplasmic and nuclear protein that was identified as a binding partner of periplakin (PPL), a component of epidermal desmosomes (DS) and the cornified envelope (CE) (Groot et al. 2004).

Kazrin has at least 5 different isoforms. Overexpression of the short isoform kazrinE stimulates the terminal differentiation of cultured human keratinocytes and is associated with a reduction in F-actin content, disruption of DS assembly, and changes in cell shape. Overexpression of activated RhoA rescues the effects on cell shape and adhesion. Conversely, knockdown of the longest isoform kazrinA impairs terminal differentiation, independently of RhoA activity (Sevilla et al. 2008a). KazrinE colocalizes with stabilized microtubules in differentiating keratinocytes (Nachat et al. 2009). All KAZN isoforms can form complexes with one another (Nachat et al. 2009), suggesting that like periplakin and envoplakin, it may form part of the cortical scaffold that integrates the actin cytoskeleton with DS (Ruhrberg et al. 1997, Kalinin et al. 2001, Groot et al. 2004). In Xenopus embryos, depletion of endogenous KAZN results in striking defects in axial elongation, muscle and notochord differentiation, and epidermal morphogenesis. These effects are believed to be due to disruption of cell-cell junctions (Sevilla et al. 2008b, Cho et al. 2010). However, mice with a knockout that removes exons 5-15 of KAZN had normal epidermal morphogenesis and homeostasis (Chhatriwala et al. 2012).

Literature references

Groot, KR., Sevilla, LM., Nishi, K., DiColandrea, T., Watt, FM. (2004). Kazrin, a novel periplakin-interacting protein associated with desmosomes and the keratinocyte plasma membrane. *J. Cell Biol., 166*, 653-9. ↗

Editions

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